Risk Factors for Pediatric Asthma
Contributions of Poverty, Race, and Urban Residence

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The Child Health Supplement to the 1988 National Interview Survey was used to examine parent-reported current asthma among a nationally representative sample of 17,110 children zero to 17 yr of age. Numerous demographic variables were analyzed for independent associations with asthma using modified stepwise logistic regression, with models including specific combinations of risk factors. Black children had higher rates of asthma than did white children in unadjusted analyses, but after controlling for multiple factors, black race was not a significant correlate of asthma (adjusted odds ratio = 0.87, 95% CI = 0.63 to 1.21). Compared with nonurban white children, urban children, both black and white, were at significantly increased risk of asthma: urban and black (adjusted OR = 1.45, 95% CI = 1.14 to 1.86), urban and white (adjusted OR = 1.22, 95% CI = 1.01 to 1.48), whereas nonurban black children were not: nonurban and black (adjusted OR = 1.15, 95% CI = 0.83 to 1.61). Similarly, compared with nonurban, nonpoor children, urban and poor (adjusted OR = 1.44, 95% CI = 1.05 to 1.95), urban and nonpoor (adjusted OR = 1.22, 95% CI = 1.004 to 1.48), urban children, both poor and nonpoor, were at significantly increased risk of asthma, whereas nonurban poor children were not: nonurban and poor (adjusted OR = 1.03, 95% CI = 0.72 to 1.48). These results suggest that the higher prevalence of asthma among black children is not due to race or to low income per se, and that all children living in an urban setting are at increased risk for asthma.

Asthma is the most common chronic illness of childhood, and despite advances in therapy, asthma prevalence, morbidity, and mortality are all increasing (1, 2). It is well recognized that, in the United States, asthma is more prevalent and more severe among black children than among white children (3–9, 10). However, it is still unclear what is causing the rise in asthma, or what is responsible for the continuing racial disparities in its prevalence and severity (11, 12).

It appears that differences in asthma prevalences between population groups are due to differential exposure to environmental factors; genetic variation alone could not account for the rise in this disease's prevalence over a few decades (13, 15). Hence, it may be that black race is merely a confounder for a set of exposures that disproportionately affect black children. In the United States, the inhabitants of impoverished inner-city areas are disproportionately black, and inner cities are the areas where asthma is worst (16–18). We hypothesize that urban residence is an independent risk factor for childhood asthma after controlling for race, poverty, and other environmental and demographic variables.

METHODS

Data Source

The 1988 Child Health Supplement (CHS) to the National Interview Survey (NHIS) was the source of data for this study (19). The NHIS uses a complex, multistage probability sampling design to provide a representative sample of the civilian, non-institutionalized population of the United States. The CHS provides information on one randomly selected child in each eligible household. The 1988 CHS is the most current complete child health supplement to the NHIS available for analysis and consists of a sample of 17,110 children zero to 17 yr of age that is representative of the approximately 64 million children in the civilian, noninstitutionalized, nonhomeless population. This database provides information related to child health issues, as well as important demographic information such as whether or not a child lived in an urban setting.

Variables

The definition of asthma employed for this study was parental report of current asthma. Parents were asked if their child ever had asthma. If they said "Yes," they were then asked if their child had asthma in the last 12 mo, and only those children so identified were categorized as having asthma.

The main independent variables investigated were race, poverty status, and urban residence. Race was categorized as black, white, or other and was based on the respondent's characterization of the child's racial background. Poverty status was determined by comparing household income to the federal poverty index for families of similar size. A child was identified as living in poverty if living below the federal poverty index, which in 1988 was $12,092 for a family of four and $16,146 for a family of six. Near-poverty was defined as living at 101 to 150% and nonpoverty as > 150% of this index. Urban residence was categorized by whether or not the child lived in the central city of a Metropolitan Statistical Area (MSA). A central city is defined as the largest city in each metropolitan area (20).

In addition to the main independent variables, several other factors were also analyzed to adjust for possible confounding. These included personal demographics and home environment characteristics such as the child's age and sex, maternal age at child's birth, and Hispanic or non-Hispanic ethnicity; geographic region of the country (Northeast, South, Midwest, and West); family characteristics such as maternal education, family size, passive smoking exposure, and single parent status; access-to-care measures, including health insurance coverage, having a recent routine medical care visit, and having a usual place for such care; and medical history items such as overall health status, birthweight, and history of allergies.

Statistical Analyses

All survey responses were weighted using the weights provided by the National Center for Health Statistics, which reflect the probability of selection, nonresponse, and poststratification adjustments (19). Bivariate analyses were performed first to identify possible predictor variables for associations with asthma, as reflected by significant chi-square statistics. These independent variables, as well as some other potentially important confounders, were then analyzed for independent associations with asthma using logistic regression analyses. In order to determine which demographic variables cause race to "drop out" of the model, asthma risk factors were added one variable at a time to a model that initially included only race as the independent variable and asthma as the dependent variable. To further elucidate the relative importance of risk factors, combinations of variables (e.g.,

urban and black versus nonurban and black) were compared. All analyses were performed with SUDAAN software (Research Triangle Institute, Research Triangle Park, NC) to account for the complex sampling design of NHIS (21).

RESULTS

Overall, 4.2% (n = 747) of children were reported as having asthma during the preceding 12 mo. As was reported in previous studies, black children were found, in unadjusted analyses, to have a significantly higher prevalence of asthma than were white children (5.1 versus 4.1%, p = 0.04, odds ratio [OR] = 1.3) (1). However, after controlling simultaneously for the multiple demographic variables mentioned above, black race was no longer a significant correlate of asthma (OR = 0.9, 95% CI = 0.63 to 1.20) (Table 1).

We thus sought to determine which of these other variables caused race to "drop out" of the model; that is to say, after controlling for what other factor(s) is race no longer a significant correlate of asthma? For the analyses shown in Table 2, the referent group utilized consisted of children who were white, not poor, and not urban. The odds ratio reflects the risk

of asthma, when compared with this referent group, for black children after controlling for each of a number of other risk factors. The first line thus describes the risk of asthma in children who are black. The second line is the odds ratio of asthma in black children after controlling only for access to regular checkups. The next line is again the odds ratio for asthma in black children, this time after controlling only for maternal education, and then so on for the other variables. As reflected by p values < 0.05, black race remained a significant correlate of asthma after controlling for each of the following factors: access to regular checkups, maternal education, maternal age, exposure to smokers in the home, Hispanic ethnicity, region, history of eczema, history of hay fever, sex, and age of child. In contrast, black race did drop out of the model when adjusting for either poverty (OR = 1.2, 95% CI = 0.98 to 1.5, p = 0.07) or urban residence (OR = 1.2, 95% CI = 0.95 to 1.4, p = 0.14). With urban residence, race, and asthma in the model, the increased risk of asthma from urban residence was reflected by a statistically significant odds ratio (OR = 1.2, 95% CI = 1.04 to 1.5, p = 0.01). With poverty, race, and asthma in the model the increased risk of asthma associated with poverty was not statistically significant (p = 0.07). Thus,

TABLE 1

RISK FACTORS FOR CURRENT ASTHMA AMONG U.S. CHILDREN ZERO TO 17 yr OF AGE*

	n	Prevalence of Current Asthma (%)	Unadjusted Odds Ratio OR	p Value	Adjusted Odds Ratio[†] OR	p Value
Sex, male	8,736	5.1	1.5	< 0.001	1.4	0.002
Age, yr						
0–5	6,209	3.2	0.7	0.001	1.6	0.02
6–11	5,092	5.1	1.1	0.24	1.3	0.01
12–17 (referent)	5,809	4.5	1.0	0.00	1.0	0.00
Race						
Black	2,924	5.1	1.3	0.04	0.9	0.38
Other	1,178	3.9	0.9	0.76	1.1	0.74
White (referent)	13,008	4.1	1.0	0.00	1.0	0.00
Hispanic	2,038	3.4	0.8	0.049	0.9	0.77
Region						
Midwest	4,522	4.2	0.9	0.54	0.8	0.13
South	5,941	4.1	0.9	0.31	0.8	0.04
West	3,424	4.3	0.9	0.60	0.9	0.47
Northeast (referent)	3,223	4.6	1.0	0.00	1.0	0.00
Urban (MSA-Central City)	5,388	5.0	1.3	0.01	1.4	0.01
Poverty status, %						
≤ 100	2,474	4.7	1.1	0.24	1.0	0.99
101–150	1,591	4.0	0.9	0.76	0.9	0.80
> 150 (referent)	13,045	4.2	1.0	0.00	1.0	0.00
Medicaid past 12 mo	1,739	6.1	1.6	0.001	1.3	0.11
Health status						
Fair/poor	443	17.2	6.9	< 0.001	6.4	< 0.001
Good	2,967	8.2	3.0	< 0.001	3.0	< 0.001
Excellent/very good (referent)	13,542	2.9	1.0	0.00	1.0	
Very low birthweight, < 1,500 g	150	7.0	1.7	0.18	0.3	0.12
Maternal education, n = 16,670						
< High school grad	3,107	3.6	0.8	0.07	0.6	0.004
College grad plus	2,722	4.3	1.0	0.68	0.9	0.53
High school grad, some college (referent)	10,791	4.5	1.0		1.0	0.00
Maternal age at child's birth < 17 yr	418	1.8	0.4	< 0.001	0.2	< 0.001
Routine care visit within 2 yr	14,175	4.6	1.5	< 0.001	1.4	0.02
Has usual place for routine care	15,143	4.5	1.8	< 0.001	1.5	0.054
Ever had eczema	1,313	11.2	3.3	< 0.001	2.5	< 0.001
Ever had hay fever	1,204	14.9	4.9	< 0.001	3.6	< 0.001

* Child Health Supplement to the 1988 NHIS.

[†] Adjusted OR obtained by adjusting for all the variables in the model simultaneously (*see* list of variables in text under METHODS) in a full logistic regression model.

Note: The following variables, which bore no significant relationship to asthma in adjusted analyses (p > 0.2), are not shown in Table 1: health insurance, low birthweight, single parent household, family size, parental smoking, behavior problem index, school failure. The p values were presented to simplify this table; p values < 0.05 are equivalent to 95% confidence intervals that do not include unity.

TABLE 2

EFFECT OF CONTROLLING FOR POTENTIAL CONFOUNDERS ON THE STATISTICAL SIGNIFICANCE OF THE RELATIONSHIP BETWEEN BLACK RACE AND ASTHMA

Risk Factors in the Model	Odds Ratio for Black Race Predicting Asthma	(95% CI)	p Value
Black race	1.3	(1.02–1.5)	0.03
Access to regular check-up + black race	1.2	(1.01–1.5)	0.03
Maternal education + black race	1.3	(1.1–1.6)	0.01
Maternal age + black race	1.3	(1.1–1.6)	0.01
Smokers in home + black race	1.3	(1.02–1.5)	0.03
Hispanic + black race	1.2	(1.01–1.5)	0.04
Region + black race	1.3	(1.05–1.6)	0.02
Poverty + black race	1.2	(0.98–1.5)	0.07*
Urban residence + black race	1.2	(0.95–1.4)	0.14*
Eczema + black race	1.3	(1.1–1.6)	< 0.01
Hay fever + black race	1.4	(1.1–1.7)	< 0.01
Health status + black race	1.0	(0.77–1.2)	0.77*
Child's sex + black race	1.3	(1.03–1.5)	0.03
Child's age + black race	1.3	(1.02–1.5)	0.03

* p Value ≥ 0.05: controlling for this factor causes race to "drop out of the model."
Note: Referent group is white, not poor, and not urban.

when only one variable at a time is combined with race in a logistic regression model, the variables that caused race to drop out of the model were poverty and urban residence. Black race also dropped out when poor health status was added in.

The results of adjusted analyses incorporating *combinations* of risk factors are displayed in Table 3.

Urban Residence and Race

Urban children, both black and white, were at significantly increased risk of asthma compared with nonurban white children, whereas nonurban black children were not at increased risk of asthma: urban and black (OR = 1.45, 95% CI = 1.14 to 1.86), urban and white (OR = 1.22, 95% CI = 1.01 to 1.48), nonurban and black (OR = 1.15, 95% CI = 0.83 to 1.61).

Urban Residence and Poverty Status

Urban children, both poor and nonpoor, were at significantly increased risk of asthma compared with nonurban, nonpoor children, whereas nonurban poor children were not at increased risk of asthma: urban and poor (OR = 1.44, 95% CI = 1.05 to 1.95), urban and nonpoor (OR = 1.22, 95% CI = 1.004 to 1.48), nonurban and poor (OR = 1.03, 95% CI = 0.72 to 1.48).

TABLE 3

URBAN STATUS TOGETHER WITH EITHER BLACK RACE OR POVERTY AS RISK FACTORS FOR CHILDHOOD ASTHMA

	Odds Ratio for Having Asthma	(95% CI)	p Value
Urban			
Black	1.4*	(1.1–1.9)	0.003
White	1.2*	(1.01–1.5)	0.04
Nonurban			
Black	1.2	(0.8–1.6)	0.38
White (referent)	—	—	—
Urban			
Poor	1.4*	(1.1–2.0)	0.02
Nonpoor	1.2*	(1.004–1.5)	0.04
Nonurban			
Poor	1.03	(0.7–1.5)	0.85
Non–poor (referent)	—	—	—

* Significantly (p < 0.05) increased odds for having asthma.

In the results shown, all ages from zero to 17 yr were grouped together. Because this could mask relevant differences in asthma risk between age groups, analyses were adjusted for age. In addition, it was noted that categorizing the data into separate age groups (0–5, 6–11, 12–17 yr) did not change the overall pattern of results with respect to race versus urban effects (not shown in table).

DISCUSSION

We found that when children are categorized by urban status, all urban children have a heightened risk of asthma, regardless of race or family income. This study employed combinations of risk factors in stepwise logistic regression to facilitate the distinction between the separate effects of race, urban status, and income after controlling for a large number of other variables in a nationally representative sample. Its results suggest that black race is a confounder for urban residence with respect to asthma risk, rather than an independent risk factor.

There is no universally accepted definition of asthma, and this complicates comparisons to other studies that used different definitions (22). We limited ourselves to a consideration of asthma prevalence, not morbidity, as it is likely that there are risk factors for morbidity measures such as hospitalization acting independently of those for prevalence. All information was derived from parental reports; such questionnaires remain the cornerstone of large asthma prevalence surveys (23). It is possible that the higher prevalence of asthma among urban, black, or poor children may be an artifact of overdiagnosis in certain subgroups of the population (24). For example, we found that low maternal age decreased the risk of doctor-diagnosed asthma, whereas access to medical care increased it (Table 1). This might suggest that there was a reporting bias with respect to diagnosis since children with older, better educated mothers who take them to the doctor more often would be more likely to have asthma diagnosed for any given level of illness severity. However, since race remained a significant correlate of asthma after controlling for maternal age or access to care, this potential bias does not appear to explain the racial disparity in childhood asthma (Table 2).

The data reported here do not prove a causal relationship, as they are based on cross-sectional survey data, but they do provide evidence that strengthens the likelihood of a link between urban environment and asthma (25). Moreover, it is quite plausible that some aspect of urban living is responsible for the increased asthma risk among black children; many factors that have been proposed as contributing to asthma (e.g., premature birth, passive smoking, substandard housing, increased time indoors, bad diet, decreased access to health care, exposure to cockroaches, etc.) tend to concentrate in urban settings (4, 13, 17, 26–28). The database used here does not allow us to investigate which aspects of living in the city may increase the risk of asthma. Some exposures that may have been of interest such as cockroach or dust mite antigen were not measured in the NHIS, and thus could not be investigated. The increased risk of asthma among urban children could make it appear, in unadjusted analyses, that black children have more asthma than white children simply because black children are much more likely than white children to live in cities (6, 29). Unfortunately, the variable for urban status used in the dataset does not allow a clear distinction between inner city, outer city, and suburb. However, if living in the inner city is the important risk factor for asthma, then a more precise definition of urban status would have yielded a more impressive odds ratio for the urban residence as a correlate of asthma; and it would have lowered the odds ratios for black race as a risk factor for asthma. Although the moderately in-

creased risk of asthma associated with urban residence is not very impressive, the finding that race does not correlate significantly with asthma suggests strongly that race is *not* an independent cause of asthma.

In addition, the finding that urban residence, rather than race, increases the risk of asthma is consistent with current theories of asthma etiology (23). Asthma appears to be a disease of modern civilization with *in utero* and early life environmental exposures leading to increased IgE-dependent hypersensitivity, and then bronchial hyperreactivity and airway inflammation (30, 31). It does not appear to be a racially linked genetic disease (32, 33). Indeed, black populations living in Africa have very low asthma prevalence, and the asthma that does occur is associated with home environmental factors related to urbanization (13, 34). These findings are also consistent with the literature on racial disparities in other aspects of health, which emphasizes that, with a few exceptions, there is little biologic meaning to the term "race" as used to distinguish "black" from "white" persons in the United States (35–39). For example, a recent study of the epidemiology of another pulmonary disease, tuberculosis, found that much of the increased risk of tuberculosis previously associated with race/ethnicity was actually accounted for by socioeconomic differences, including characteristics of living conditions (40).

Racial categories are too broad to be meaningful, thus important differences within racial groups may be masked. "White" people are not all the same, nor are all "black" people. In addition, from an epidemiologic perspective, distinctions between race and ethnicity are unclear. Hence, categorizing all Mexicans, Puerto Ricans, and Argentineans together as "Hispanic" is also imprecise. Associating diseases with racial or ethnic status, when these are imprecise risk markers and not risk factors, leads to spurious biologic connections (37).

When searching for disease etiology, it is important to look at differences between populations and not just between individuals (41). If asthma is related to some aspect of indoor air pollution (for example), and if everyone in a given population is exposed to that combination of pollutants, then the distribution of cases is wholly determined by individual susceptibility, and it would seem that asthma is a genetic disease even when it is not. Thus it is important to have a broad perspective when looking for causes of asthma.

The International Study of Asthma and Allergies in Childhood (ISAAC) is an on-going international study of children 13 to 14 yr of age using an extensively validated written and video questionnaire (42). It revealed very striking differences in the prevalence of asthma worldwide, with the highest prevalence about 20 times higher than the lowest (range, 1.6 to 36.8%). The United States is among the high prevalence countries. The worldwide variations in rates suggest that environmental factors are critical to the development of asthma.

Immigrants from countries with a low asthma prevalence such as India or Malaysia who now live in countries with a high asthma prevalence such as England or Australia, tend, over the course of a few decades, to develop asthma at rates similar to those in the population they have joined (43). Black African countries such as Ethiopia and Nigeria tend to have a very low asthma prevalence (13). In Kenya, another black African country, pediatric asthma has been found to be associated with home environmental factors related to urbanization and industrialization, especially as they affect indoor air in the child's bedroom (44). Thus comparisons of black populations show that asthma is not evenly distributed throughout all black people and that its presence appears to be related more to environment than to "race."

Comparisons over time reveal that the prevalence of asthma in both developing and developed countries, including many parts of Africa, seems to have increased considerably; this phenomenon is more plausibly explained by urbanization and the accompanying changes in lifestyle than by changes in genes (31). Genetic variation alone could not account for the rise in this disease's prevalence over a few decades (13–15). Asthma appears to be a disease of modern civilization, not a racially linked genetic disease (23, 30, 31).

We used a variable based on family income as a proxy for SES, but it has been demonstrated recently that income is not an accurate yardstick for comparing SES between black and white Americans: for example, black people tend to have much less accumulated wealth than white people, even at similar incomes (45). As a result, using income to adjust for SES may not adequately control for true black/white SES differences. Such inadequate controlling for SES could make it appear that black "race" is a risk factor for disease when it is not. This bias makes it all the more remarkable that we do indeed find race "dropping out" of the model in this study.

The United States is one of the few countries in the world where asthma appears to be related to poverty (31). City living is a condition that is associated with poverty in the United States but that has historically been associated with wealth in other countries, and in the past asthma was associated with wealth rather than poverty (23, 46). Thus it is plausible that the relationship between urban living and asthma could be confounded by poverty, as these results suggest. Using other means to adjust for SES, e.g., parental occupation, and for the concentration of poverty in inner-city areas may help to further elucidate the relationship between these factors and asthma (47).

To distinguish between the separate effects of race, urban status, and poverty on the risk of asthma, this study employed stepwise logistic regression to examine combinations of risk factors using data from the National Health Interview Survey. Although some previous reports have found that race remains a significant correlate of asthma prevalence after controlling for SES and environmental variables, those studies were not able to compare urban with nonurban status as we did here, because their populations were limited to only urban children or to only nonurban children (5, 48, 49). Other reports found, as we did, that race is not an independent predictor of asthma, but those studies did not specifically examine which variable black race was a confounder for (29, 50). Black race dropped out of the model with the addition of health status. However, this does not imply that black race is a confounder for health status since overall health status is affected by asthma (51).

Conclusions

These results suggest that the disturbingly higher prevalence of asthma among black children in the United States is not attributable to race. All children living in an urban setting, regardless of race or income, are at increased risk of asthma. These data are consistent with other findings which indicate that asthma should not be thought of as an irremediable genetic problem of some population subgroups, but rather as a consequence of exposure to a modern urban environment. This would imply that black children have more asthma because they are more likely to live in urban, poor neighborhoods. Thus, investigating specific urban environmental factors that contribute to asthma should lead to insights that will help to correct persisting racial disparities in asthma, and reduce the prevalence and severity of this illness for all children.

References

1. Weitzman, M., S. L. Gortmaker, A. M. Sobol, and J. M. Perrin. 1992. Recent trends in the prevalence and severity of childhood asthma. *J.A.M.A.* 268:2673–2677.

2. Weiss, K. B., P. J. Gergen, and D. K. Wagener. 1993. Breathing better or wheezing worse? The changing epidemiology of asthma morbidity and mortality. *Annu. Rev. Public Health* 14:491–513.

3. Newacheck, P. W., P. P. Budetti, and N. Halfon. 1986. Trends in activity-limiting chronic conditions among children. *Am. J. Public Health* 76:178–184.

4. Evans, R., III. 1992. Asthma among minority children: a growing problem. *Chest* 101:368S–371S.

5. Cunningham, J., D. W. Dockery, and F. E. Speizer. 1996. Race, asthma, and persistent wheeze in Philadelphia schoolchildren. *Am. J. Public Health* 86:1406–1409.

6. Crain, E.F., K. B. Weiss, P. E. Bijur, M. Hersh, L. Westbrook, and R. E. Stein. 1994. An estimate of the prevalence of asthma and wheezing among inner-city children. *Pediatrics* 94:356–362.

7. Weiss, K. B., and D. K. Wagener. 1990. Changing patterns of asthma mortality: identifying target populations at high risk. *J.A.M.A.* 264:1683–1687.

8. Schwartz, J., D. Gold, D. W. Dockery, S. T. Weiss, and F. E. Speizer. 1990. Predictors of asthma and persistent wheeze in a national sample of children in the United States: association with social class, perinatal events, and race. *Am. Rev. Respir. Dis.* 142:555–562.

9. Weitzman, M., S. Gortmaker, and A. Sobol. 1990. Racial, social, and environmental risks for childhood asthma. *Am. J. Dis. Child.* 144:1189–1194.

10. National Asthma Education and Prevention Program. 1997. Expert Panel Report 2: Guidelines for the Diagnosis and Management of Asthma. National Heart, Lung, and Blood Institute, Bethesda, MD. NIH Publication No. 97-4051.

11. Nsouli, T. M. 1999. Inner-city disadvantaged populations and asthma prevalence, morbidity, and mortality. *Ann. Allergy Asthma Immunol.* 82:2–4.

12. Mitka, M. 1999. Why the rise in asthma? New insight, few answers. *J.A.M.A.* 2171–2172.

13. Platts-Mills, T. A., and M. C. Carter. 1997. Asthma and indoor exposure to allergens. *N. Engl. J. Med.* 336:1382–1384.

14. Mellis, CM. 1994. Childhood asthma appears to be increasing: but how good is the evidence? *J. Paediatr. Child Health* 30:387–388.

15. Drazen, J. M., and E. K. Silverman. 1997. Genetics of asthma: conference summary. *Am. J. Respir. Crit. Care Med.* 156:S69–S71.

16. Weiss, K. B., P. J. Gergen, and V. F. Crain. 1992. Inner-city asthma: the epidemiology of an emerging public health concern. *Chest* 101:362S–367S.

17. Kattan, M., H. Mitchell, P. Eggleston, P. Gergen, E. Crain, S. Redline, K. Weiss, R. Evans, 3rd, R. Kaslow, C. Kercsmar, F. Leickly, F. Malveaux, and H. J. Wedner. 1997. Characteristics of inner-city children with asthma: the national cooperative inner-city asthma study. *Pediatr. Pulmonol.* 24(4):253–262.

18. Grant, E. N., R. Wagner, and K. B. Weiss. 1999. Observations on emerging patterns of asthma in our society. *J. Allergy Clinical Immunol.* 104:51–59.

19. National Center for Health Statistics. 1982. Current estimates from the National Health Interview Survey: United States, 1981. *Vital Health Stat. 10.* 14:1–106.

20. U.S. Bureau of the Census. 1988. Current Population Reports, Series P-26: Local Population Estimates Government Printing Office, Washington, DC.

21. Shah, B. V., B. G. Barnwell, and G. S. Bieler. 1997. SUDAAN User's Manual, Release 7.5. Research Triangle Institute, Research Triangle Park, N.C.

22. Silverman, M., and N. Wilson. 1997. Asthma: time for a change of name. *Arch. Dis. Child.* 77:62–65.

23. Pearce, N., R. Beasley, C. Burgess, and J. Crane. 1998. Asthma Epidemiology: Principles and Methods. Oxford University Press, New York.

24. Gergen, P. J. 1996. Social class and asthma: distinguishing between the disease and the diagnosis. *Am. J. Public Health* 86:1361–1362.

25. Hill, A. B. 1965. The environment and disease: association or causation. *Proc. R. Soc. Med.* 58:295–300.

26. Lang, D. M., and M. Polansky. 1994. Patterns of asthma mortality in Philadelphia from 1969–1991. *N. Engl. J. Med.* 331:1542–1546.

27. Apter, A. J., S. T. Reisine, G. Affleck, E. Barrows, and R. L. ZuWallack. 1998. Adherence with twice-daily dosing of inhaled steroids. *Am. J. Respir. Crit. Care Med.* 157:1810–1817.

28. Malveaux, F. J., and S. A. Fletcher-Vincent. 1995. Environmental risk factors of childhood asthma in urban centers. *Environ. Health Perspect.* 103:59–62.

29. Gergen, P. J., D. I. Mullally, and R. Evans. 1988. National survey of prevalence of asthma among children in the United States, 1976–1980. *Pediatrics* 81:1–7.

30. Holgate, S. T. 1998. Asthma and allergy: disorders of civilization? *Q. J. Med.* 91:171–184.

31. Platts-Mills, T. A. E. 1997. Asthma among inner city children. *Pediatr. Pulmonol.* 24:231–233.

32. Smith, J. M., R. J. Cadoret, T. L. Burns, and E. P. Troughton. 1998. Asthma and allergic rhinitis in adoptees and their adoptive parents. *Ann. Allergy Asthma Immunol.* 81:135–139.

33. Sanford, A., T. Weir, and P. Pare. 1996. The genetics of asthma. *Am. J. Respir. Crit. Care Med.* 153:1749–1765.

34. Mohamed, N., L. Ng'ang'a, J. Odhiambo, J. Nyamwaya, and R. Menzies. 1995. Home environment and asthma in Kenyan schoolchildren: a case-control study. *Thorax* 50:74–78.

35. Foster, H. W., Jr. 1997. The enigma of low birth weight and race. *N. Engl. J. Med.* 337:1232–1233.

36. Navarro, V. 1990. Race or class versus race and class: mortality differentials in the United States. *Lancet* 336:1238–1240.

37. Centers for Disease Control and Prevention. 1993. Use of race and ethnicity in public health surveillance: summary of the CDC/ATSDR Workshop. *M.M.W.R.* 42:1–16.

38. Angell, M. 1993. Privilege and health: what is the connection? *N. Engl. J. Med.* 329:126–127.

39. Montgomery, L. E., J. L. Kiely, and G. Pappas. 1996. The effects of poverty, race, and family structure on US children's health: data from the NHIS, 1978 through 1980 and 1989 through 1991. *Am. J. Public Health* 86:1401–1405.

40. Cantwell, M. F., M. T. McKenna, E. McCray, and I. M. Onorato. 1998. Tuberculosis and race/ethnicity in the United States: impact of socio-economic status. *Am. J. Respir. Crit. Care Med.* 159:1016–1020.

41. Rose, G. 1985. Sick individuals in sick populations. *Int. J. Epidemiol.* 14:32–38.

42. The International Study of Asthma and Allergies in Childhood. 1998. Worldwide variation in prevalence of symptoms of asthma, allergic rhino conjunctivitis, and atopic eczema: ISAAC Steering Committee. *Lancet* 351:1225–1232.

43. Leung, R. 1996. Asthma and migration. *Respirology* 1:123–126.

44. Mohamed, N., L. Ng'ang'a, J. Odhiambo, J. Nyamwaya, and R. Menzies. 1995. Home environment and asthma in Kenyan schoolchildren: a case-control study. *Thorax* 50:74–78.

45. Kaufman, J. S., R. S. Cooper, and D. L. McGee. 1997. Socioeconomic status and health in blacks and whites: the problem of residual confounding and the resiliency of race. *Epidemiology* 8:621–628.

46. McKeown, T. 1988. The Origin of Human Disease. Basil Blackwell Inc., Oxford, UK.

47. Ernst, P., K. Demissie, L. Joseph, U. Locher, and M. Becklake. 1995. Socioeconomic status and indicators of asthma in children. *Am. J. Respir. Crit. Care Med.* 152:570–575.

48. Gold, D. R., A. Rotnitzky, A. I. Damokosh, J. H. Ware, F. E. Speizer, B. G. Ferris, Jr., and D. W. Dockery. 1993. Race and gender differences in respiratory illness prevalence and their relationship to environmental exposures in children 7 to 14 years of age. *Am. Rev. Respir. Dis.* 148:10–18.

49. Nelson, D. A., C. C. Johnson, G. W. Divine, C. Strauchman, C. L. Joseph, and D. R. Ownby. 1997. Ethnic differences in the prevalence of asthma in middle class children. *Ann. Allergy Asthma Immunol.* 78:21–26.

50. Wissow, L. S., M. Gittlesohn, M. Szklo, B. Starfield, and M. Mussman. 1988. Poverty, race, and hospitalization for childhood asthma. *Am. J. Public Health* 78:777–782.

51. Forrest, C. B., B. Starfield, A. Riley, and M. Kang. 1997. The impact of asthma on health status of adolescents. *Pediatrics* 99:E1.